biotics. Polk and Lopez-Mayer²⁵ gave cephaloridine alone in the same way, and their practice was followed by Evans and Pollock.²⁷ Campbell²⁸ relied on large doses of penicillin alone, and Stokes et al19 reported encouraging preliminary results from two doses of a combination of lincomycin with either gentamicin or tobramycin. This form of prophylaxis is logical, innocuous, since side effects are unlikely from so few doses, and evidently effective. And it has wide applications: the more specialised uses of antibiotics in surgery, notably the prevention of infection in burns, cannot be discussed here.

Among other applications in surgery are the prevention of urinary tract infections, referred to above, and the local application of antibiotics in wounds. This is another relatively innocuous use which is to be recommended in so far as it is proved effective. This type of use and the systemic administration of only a few doses are much to be preferred to the indiscriminate and damaging systemic treatment continued for days which was in vogue 20 years ago.

I thank H F Wood and the editor of Annals of Internal Medicine for permission to reproduce table I and the editor of British Journal of Obstetrics and Gynaecology for permission to include in table II data first published in that journal.

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Hospital Topics

Management of uraemic pericarditis

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British Medical Journal, 1975, 4, 564-566

Summary

Of 250 patients undergoing haemodialysis from 1967 to 1974 17 presented with uraemic pericarditis. Seven of these patients who had been transferred early enough to peritoneal dialysis treatment were cured without pericardiectomy (mean survival 18 months (range 6-36); no deaths). Only one patient was cured from his pericarditis by "aggressive haemodialysis." In seven out of 10 patients treated with haemodialysis, pericardiectomy finally had to be performed because of pericardial tamponade (postoperative survival 20 months (range 8-36); one death). Two patients died from pericardial tamponade before surgery.

In patients with evidence of uraemic pericarditis

frequent peritoneal dialysis with high fluid withdrawal is the treatment of choice, but in cardiac tamponade pericardiectomy should follow a preoperative pericardiocentesis with limited fluid aspiration.

Of possible significance in the aetiology of pericarditis were the findings that 10 of the 17 patients had hypertension with cardiac enlargement and that 14 presented with evidence of underdialysis, possibly due to the reuse of dialysis components.

Introduction

The first report on pericarditis in patients with kidney disease dates back to 1836: Bright1 observed pericarditis together with pleural and peritoneal effusion in 100 necropsy cases "connected with albuminous urine." The high frequency of pericarditis in uraemia may be shown by necropsy findings from the period before the introduction of dialysis treatment. Various workers 7 10 14 16 observed uraemic pericarditis in 20-50% of the patients who died from uraemia.

The frequency of uraemic pericarditis has been reduced to less than 15% by the introduction of dialysis. 4 13 17 A statistical investigation by the European Dialysis and Transplant Association, which registered 3800 deaths among patients on haemo-

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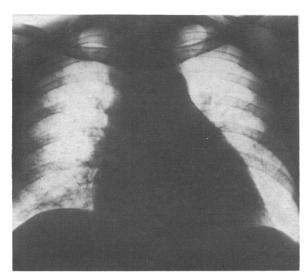
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dialysis up to December 1972, showed that haemorrhagic pericarditis was the cause of death in 5.2%. According to our experience this number may be reduced to 1% by strict adherence to the regimen proposed here.

Patients and symptoms

Of 250 patients undergoing haemodialysis from 1967 to 1974 17 presented with uraemic pericarditis. The clinical signs were pericardial friction rub in 10, cardiac pain in nine, and fever in eight. Five patients presented with acute pericardial tamponade. One of the most important differential diagnostic signs of acute pericardial tamponade was the absence of pulmonary congestion despite acute cardiac enlargement. The contours of the enlarged heart (see fig) on the x-ray film were more often rounded than triangular.



Enlargement of heart due to acute pericardial effusion. Contours of heart on x-ray film were more rounded than triangular

Treatment

In uncomplicated cases of uraemic pericarditis the treatment of choice proved to be frequent peritoneal dialysis (2-3 \times 24h/week) with no heparin administration and high fluid withdrawal. Cure was achieved without pericardiectomy in all 7 patients who had been transferred early enough (within one week of onset of friction rub in four patients and immediately after the diagnosis of a pericardial effusion in three) to peritoneal dialysis. At the time of writing the mean survival time in these patients, none of whom died, was 18 months (range 6-36 months). In all cases the Tenckhoff indwelling catheter was used with success. Pericardial friction rub disappeared and the size of the heart returned to normal after four to eight weeks.

Only one patient was cured from his pericarditis by "aggressive haemodialysis" (4×8 h/week). The other nine patients treated with haemodialysis finally developed evidence of pericardial tamponade. One patient died after fenestration of the pericardial sac as a result of pericardial abscess formation, and two died from pericardial tamponade before surgery. In the other six patients two-thirds of the pericardium were removed through a thoracotomy via the left fifth interspace. The mean postoperative survival at the time of writing in these patients was 20 months (range 8-38 months). One patient died from hypertensive heart disease 30 months after pericardiectomy as a result of poor co-operation.

Possible aetiological factors in uraemic pericarditis

Seven of the patients with pericarditis were among 34 patients undergoing home dialysis using the same type of equipment and following approximately the same time schedule (Multipoint-Kiil, 3×8 h/week). Pericarditis occurred more often in those of the 34 who had a high blood pressure (table I). Hypertension in these patients

was mostly the result of undisciplined dialysis and fluid intake. A similar conclusion may be drawn from an analysis of the relation between the serum creatinine concentration after the longest dialysisfree interval and the frequency of pericarditis. In 14 out of the 17 patients with pericarditis the creatinine levels were higher than 1061 $\mu mol/l$ (12 mg/100 ml) for a long time before the first clinical symptoms appeared. It is of particular interest that these patients had been treated with either reused flatbeds or ineffective peritoneal dialysis. So far we have seen no cases of pericarditis in patients on dialysis in (3 hospital \times 8 h/week) where the flatbeds are only used once.

The relation between infection or fever and pericarditis (table II) was less convincing, though nine of the 17 patients presented with some kind of infection during the period before clinical diagnosis. Only five of these patients had fever before diagnosis, but a further four patients developed fever on the day of diagnosis or some days later.

TABLE I—Relation between hypertension and pericarditis in 34 home dialysis patients

	Hypertension (≥160/100 mm Hg)	Normotension (<160/100 mm Hg)
No of patients	10	24
No with pericarditis	5 (50%)	2 (8·3%)

TABLE II—Infection or fever in 17 patients with uraemic pericarditis

Case No	Infection		Duration of fever
	Type	Duration	before diagnosis (days)
1	Hepatitis	6 Weeks	20
2	Influenza	2 Weeks	14
3	Cold	2 Weeks	14
4	Peritonitis	2 Weeks	7
5	Bacteraemia		7
6	Hepatitis	10 Days	0*
7	Endometritis	2 Weeks	0*
8, 9			0*
10,	Cold	2 Weeks	No fever
īĭ	Cold	3 Weeks	No fever
12-17			No fever

^{*}Developed fever on or after day of diagnosis.

Discussion

On the basis of our observations and those of others some major points about the prophylaxis and treatment of uraemic pericarditis should be taken into consideration. Firstly, cardiac dilatation as a result of high pressure load or volume load increases the risk of pericarditis.

Secondly, reuse of dialysis components may be one of the factors causing pericarditis, not because of the increased risk of bacterial invasion but mainly because of the decreasing efficiency of dialysis with each reuse. This conclusion would accord with the results of Fürst et al, who observed in patients with pericarditis an accumulation of middle molecule peptides like those found in uraemic patients not undergoing dialysis. As a rule, therefore, serum creatinine levels after the longest dialysisfree interval should generally not be higher than 1061 µmol/l (12 mg/100 ml).

Thirdly, the clinician should pay more attention to the pericardial sac in patients with any kind of infection or fever who are undergoing dialysis. Besides the well-known signs of acute pericardial tamponade one of the most important differential diagnostic signs is the absence of pulmonary congestion despite acute cardiac enlargement. (The absence of pulmonary congestion may be explained by the different endogenous elasticity of the two ventricles. The filling of the thick-walled left ventricle with blood is less hampered by an increased pericardial pressure than the filling of the thin-walled right ventricle. Thus, even in the presence of high pericardial pressure, the left ventricular stroke output may be higher than that of the right ventricle.)

Fourthly, in patients with evidence of pericarditis frequent peritoneal dialysis with high fluid withdrawal should be started immediately, using a Tenckhoff catheter. This treatment should be continued for a further three weeks after the size and shape of the heart have returned to normal and the pericardial friction rub has disappeared.

Fifthly, in cardiac tamponade pericardiectomy should follow a preoperative pericardiocentesis with limited fluid aspiration to relieve the cardiac embarassment. Extensive pericardiectomy has distinct advantages over pericardial fenestration, because recurrence of tamponade from closure of the window is avoided.15

Lastly, high-dose systemic steroid administration is of little help because of steroid-induced complications such as increased protein catabolism,12 peptic ulcer, purulent peritonitis, and a rather high incidence of adhesive pericarditis.4 5 The beneficial effect of local administration of steroids is questionable. Only one successful treatment with a follow-up under haemodialysis of more than three months has been reported; in the other patients the cause of uraemic pericarditis was eliminated by successful kidney transplantation shortly after the pericardial rinsing.3

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Inflation down under

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British Medical Journal, 1975, 4, 566-567

The financial crunch has come here, just as it has in Britain. It is probably not so severe, and it may be that the recession is only in its early stages. It is hoped that the world economy will soon recover and so help Australia to avoid the worst effects of inflation and unemployment which have for so long afflicted the United Kingdom. Australia has such vast quantities of raw materials and produce that if only there are enough foreign buyers she can quickly recover. All the developed countries are caught in the same vicious spiral, all being dependent on the others to break out of it.

The history of this particular disease is becoming clearer. After a long period of uninterrupted conservative government the electorate decides on a change and feels that it has developed a heart and a care for the downtrodden, who must be identified and helped. A socialist government comes in, pledged to righting wrongs which have not been recognised by its predecessors. Money is poured into heart-warming social, scientific, and medical projects. The caring revolution is underway and life will be improved for all and become more civilised. Bureaucracy also increases to administer this vast social change. The schools and the universities, and other institutions of tertiary education, must look to their curricula, change their courses, and cross interdisciplinary boundaries, so that people of high calibre shall be produced to take advantage of and feed the burgeoning technology. There is a bonanza for those in medicine, science, and sociology. Universities and tertiary education in general expand their staffs. Students gain entry to these places almost without let or hindrance, though they must show some academic capability. The faculties struggle and vie with one another to expand their courses and make them more meaningful for the modern world. In this they are helped and kicked by a vociferous student body, who know what the present world is about, whereas the older generation, and especially their teachers, do not. There is a happy feeling of expansion, and whatever is wanted will be provided for. And so it is—up to a point.

The liberation from the old ways breeds a way of thinking that it is right that all should have access to higher education, and that research into anything and everything will make life better. There is pique when a particular project or educational change or reform is refused by denying it funds. When the funds are fairly easy to come by such refusals are made by value judgment of the potential worth of the research But quite quickly some realise, and it soon becomes apparent to all, that the revolution has outreached itself. It is not only a question of willing the means, it is a question too of finding them. Evidently the caring revolution is utterly dependent on a sound economic basis of society.

The means to support this new type of society can be found by borrowing, printing more money, increasing taxation. These are only temporary for the bills must ultimately be paid, and the ordinary person, the taxpayer, does not like that. He wants the caring society, but does not want to pay for it. Moreover, he is an elector and demands that the government should do something about it, reduce inflation and unemployment. The government has to think about the bases of society, and establish in its own mind what need is primary and what is secondary or even tertiary. It dare not make this philosophy

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